

Correspondence.

THE MORTALITY OF APPENDICITIS.

SIR,—The excellent and thoughtful papers of Mr. Joseph E. Adams and of Mr. James Sherren (*JOURNAL*, April 18th, pp. 723 and 727) once more direct attention to this subject. Their information is so complete and their arguments so moderately stated that I should hesitate to intervene were it not to join in the note of warning which you so promptly, and I think wisely, sound in your leader in the same issue.

All surgeons must know that many cases of appendicitis spontaneously recover, even when complicated by peritonitis or abscess, but that many only do so after running the gauntlet of a long and dangerous illness. Even when under observation and control in hospitals disappointments occur, and they frequently happen when attempts are made to treat patients on conservative lines in their own homes.

I have watched the progress of the surgery of appendicitis in this district for over a quarter of a century, and during this time I have experienced the results of the recurring advocacy of the delayed operation. Every time this plan has been written about in the medical press and generally discussed, it has almost at once been followed by an increased mortality. The conduct of the case on conservative lines is not reserved for the operating surgeon who ought to be responsible, but is undertaken by another section of the profession, and in the hope of avoiding operation is eagerly accepted by a not inconsiderable section of the public. Thus delay is lightly countenanced; some cases are allowed to develop general peritonitis, many abscesses occur, many fortunately localize or remain so, but too frequently complications and a lengthier stay in hospital follow, and in the long run the mortality always rises.

The following is a record of my personal operations up to the end of 1924.

Mortality of Appendicitis: Personal Experience.

	Cases.	Deaths.	Percentage.
Group 1.—Acute appendicitis without peritonitis	102	0	—
Group 2.—Acute appendicitis with localized peritonitis	366	4	1.09
Group 3.—Acute appendicitis with flank or pelvic peritonitis or both	187	15	8.02
Group 4.—Acute appendicitis with diffuse peritonitis	89	26	29.21
Group 5.—Appendicitis with residual abscess	62	5	8.06
Group 6.—Appendicitis with primary localized abscess	373	10	2.64
Group 7.—Appendicitis—interval removals	7.5	3	0.4
Group 8.—Appendicitis with primary complications	19	8	42.1
	1,938	71	3.65

All the cases for which I have been responsible are included. Group 8 comprises complications like intestinal obstruction associated with acute appendicitis, pylophlebitis, and so forth, so that the percentage mortality must needs be high, but these cases are included for the sake of completeness.

I believe that, however the figures are used, they show that the mortality rises with the march of pathological processes. The latter do not attend on the clock, and a recognition of this fact long ago made me give up a classification of cases based merely on lapse of time. One patient may show an unlimited and severe peritonitis in less than twenty-four hours, whereas in another the inflammation may always remain localized.

Recognizing my responsibility as a teacher, I would strongly urge that every case of appendicitis should be operated upon while the disease is active. Only in a few fulminating cases the surgeon must know "no night, no day," but there should be no undue delay, though the surgeon of experience may recognize that the case may safely await his convenience from, say, late at night to the next morning, or from one day to another, and so forth. There are also cases in which some delay may be an

advantage—to combat toxæmia, to allow the effect of drugs to pass off, to move the patient to better surroundings, etc.; but these considerations do not affect the general rule that *an attack of appendicitis should be cut short by operation*. The scope of the operation is a very different matter, and that must vary with the pathological conditions present and with the experience of the surgeon. A timely incision under a local anaesthetic, or the puncture of a pelvic abscess, may each save the life of a patient, so that the appendix may be removed later to prevent so serious a risk recurring. As a matter of fact, in over 95 per cent. of my own cases the appendix has been removed at the one and only operation.

But appendicitis must surely always have a mortality—and mainly the mortality of the occasional difficulty in diagnosis. Last year I had 85 cases without a death. So far this year there have been two deaths—one because some bronchial trouble associated with one of the exanthemata in a child led us to adopt the policy of the delayed operation; and one because the patient himself delayed too long before seeking the advice of his doctor. And even interval operations have some mortality.—I am, etc.,

Newcastle-upon-Tyne, April 23rd.

G. GREY TURNER.

RECENT ADVANCES IN THE STUDY OF CARDIO VASCULAR DISEASE.

SIR,—Dr. Harrington Sainsbury, in his letter (*BRITISH MEDICAL JOURNAL*, March 28th, p. 633), questions whether the modern instrumental methods have led to a real advance in the study of cardio-vascular disease or if they have merely led to changes in our points of view. I am afraid this must remain a matter of opinion.

In his comments upon my reference to the effect of Sir James Mackenzie's work upon clinical cardiology, Dr. Sainsbury discusses some defects of the polygraph and restates his views regarding the uncertainty of the physiological interpretation of the venous pulse tracing as recorded by this instrument. That physiologists do not consider the present interpretation of the various waves upon a phlebogram as settled is evidenced by Professor Barry's recent work, in which he records this pulsation by an optical method.

In consideration of the significance of venous pulse records due attention must be given to the experimental evidence brought forward by those who are engaged in the investigation, by modern methods, of the true time relationship of the various events in these records. Dr. Sainsbury appeals to those teaching to look into this matter with an open mind, and I feel certain his views will receive full consideration.—I am, etc.,

Belfast, April 20th.

J. E. MACILWAINE.

TREATMENT OF DIABETES BY RAW FRESH GLAND (PANCREAS).

SIR,—In a recent communication to the *BRITISH MEDICAL JOURNAL* (March 14th, p. 503) Dr. T. J. Hollins stated that when a patient with diabetes mellitus was given raw pancreas by mouth the sugar disappeared from the urine, and, further, that this line of treatment did not cause any symptoms of overdosage. Dr. W. Dunn (April 4th, p. 680) stated that he had had good results on one patient, but Dr. G. A. Harrison (April 18th, p. 760) failed to confirm these observations on two patients, who had been under treatment for some time. This subject is of importance, since a patient would much rather take a medicine by mouth than have it injected every day. An attempt was therefore made to confirm Dr. Hollins's work.

The patient, a girl aged 14, had been under treatment for the past two years, but her sugar tolerance had decreased, chiefly owing to the unsatisfactory conditions in the home. The dose of insulin was increased rapidly from 5 units to 12 units in the morning and another 8 units at night. After three weeks the blood sugar at 10 a.m., before the insulin injection, was normal, and the dose of insulin was reduced to 8 units in the morning and 6 units at night, owing to some slight symptoms of hypoglycaemia. The blood sugar for three days before the experiment varied from 0.12 to 0.14 per cent. The diet was kept constant at sugar

16 grams, protein 82 grams, fat 134 grams, caloric value 1,600. Weight of patient, 68 lb. The pancreas was supplied by the British Drug Houses, Ltd., and I wish to thank them for their courtesy in sending it. It was kept in an ice-chest, since insulin is not destroyed under these conditions. One ounce was eaten at 10 a.m. each day with salad, and the girl did not object to it.

Blood Sugar.			
	10 a.m.		4 p.m.
April 2nd ...	0.125 per cent.	0.14 per cent.
„ 3rd ...	0.15 „	0.21 „
„ 4th ...	0.215 „	0.225 „

Sugar appeared in the urine on the afternoon of the second day and was present in every specimen passed on the third day. The experiment was then stopped and insulin given for the next two days. Since the frozen raw pancreas had failed to maintain the blood sugar within normal limits, it was conceivable that some change had taken place as the result of the freezing. The pancreas was therefore sent direct from the slaughterhouse (packed in a little ice) and was given to the patient as soon as it arrived. This was a little inconvenient as the time of arrival varied from 1 p.m. to 4 p.m.

On the first two days 2 oz. were given and 4 oz. on the third day.

Blood Sugar.			
	10 a.m.		4 p.m.
April 6th ...	0.14 per cent.	0.13 per cent.
„ 7th ...	0.175 „	0.19 „
„ 8th ...	0.18 „	0.215 „

Sugar appeared in the urine on the afternoon of April 7th, but was absent on the morning of the third day, although in the whole twenty-four hours 12 grams of sugar were excreted. In order to complete the experiment insulin was given for the next few days, and in the following three days neither insulin nor raw pancreas was given, so as to demonstrate the effect of the pancreas.

Blood Sugar.			
	10 a.m.		4 p.m.
April 14th ...	0.115 per cent.	0.12 per cent.
„ 15th ...	0.145 „	0.145 „
„ 16th ...	0.14 „	0.15 „
„ 17th ...	0.16 „	„

Insulin started.
April 18th ... 0.115 per cent.

The experiments on this patient, together with those reported by Dr. Harrison, seem to show quite clearly that raw pancreas, when eaten, does not have any effect in lowering the blood sugar of the patient who requires insulin to maintain it at the normal level. In this case, the amount of insulin required was only 14 units, but in both experiments the blood sugar began to rise about twenty-four hours after the insulin had been stopped. When neither pancreas nor insulin was given, the blood sugar did not rise as high as when the pancreas was given, but this may be due to the extra food, which was eaten in the form of pancreas, since no reduction was made in the other constituents of the diet.

These experiments do not hold out any hope that raw pancreas can be of any assistance to the patient with true diabetes mellitus who needs insulin.—I am, etc.,

London, W.1, April 25th.

GEORGE GRAHAM.

SIR,—Dr. G. A. Harrison in his letter (April 18th, p. 760) twice mentions that “the dose of insulin remained constant.” Dr. Hollins (March 14th, p. 503), Dr. Young (March 28th, p. 632), and also Dr. Dunn (April 4th, p. 680), all three explicitly state that no insulin was being used. What becomes of Dr. Harrison's argument?—I am, etc.,

EDWARD B. FFENELL, M.D.

Hayle, Cornwall, April 24th.

INFLUENZAL MYOCARDITIS.

SIR,—During the recent influenzal outbreak I was struck by the fact that in almost every case, although the attack itself was often very mild, the convalescence was prolonged, and characterized by symptoms and signs which pointed to invasion of the myocardium by the toxin and the production of incapacity for work for an average period of four to six weeks in many cases.

The typical history given by patients was that the attack was so slight that they did not think it necessary to consult a doctor, but, following the attack, they were seized with

giddiness, faintness, breathlessness, and exhaustion. An examination of the heart revealed a somewhat rapid pulse, 80 to 90, of low tension, and irregular, and in the majority of cases there was a definite displacement of the apex beat 1/2 in. to 1 in. outwards, showing slight dilatation. Auscultation generally revealed a tic-tac rhythm, and occasionally a systolic murmur developed at the apex, but disappeared when the apex travelled in again, and the symptoms of cardiac insufficiency disappeared. Some cases were characterized by bouts of simple tachycardia and trembling of the limbs, the patient comparing his sensations to the staggering gait of a drunken person. The complexion in the majority of cases was sallow and the eyes sunken, and the cardiac symptoms were accompanied by a loss of flesh and anorexia. The symptoms generally took two months to disappear completely, and in cases of pre-existing heart trouble a longer period was required.

I find that the administration of salicin in 20-grain doses every two hours for the first twelve hours and then every three hours for the next twelve hours, as advised by E. B. Turner, has a valuable effect in protecting the heart, and should be given with the regularity of salicylates in acute rheumatism.—I am, etc.,

Stafford, April 25th.

C. JOYNER, M.A., M.B., Ch.B.

SLOW HEART.

SIR,—The *Observer* for April 26th, in a long article by a special correspondent on Nurmi, “the world's greatest athlete,” mentions that “Nurmi's heart, even under stress, beats only 44 to 49 times to the minute.” The article also recalls that at Stockholm, on August 23rd, 1923, Nurmi set up a new world's record for the mile of 4 min. 10½ sec., and that at the Paris Olympiad last year he won the 1,500 metres, 5,000 metres, 3,000 metres team race, and the 10,000 metres cross-country race. As many of your readers will know, Nurmi is still astonishing the athletic world. But it is the mention of his heart rate (44 to 49) which prompts this letter, particularly as a week ago I saw a lady, aged 30 years, whose pulse was 27 and next day 28 to the minute. She was not complaining of her health, and I had only felt her pulse because, when I said the pulse of her son, whom I was attending, was slow (50) she said it might be a family complaint.

In a long experience I can remember only one case of such very slow pulse unassociated with complaint of ill health, and that was one of a gentleman who died in his 80th year. His pulse, when he considered himself feeling well, used to be 28 a minute. He had the slow pulse during the last fourteen years of his life—I did not know him before—although during the greater part of that time he was active and took considerable walking exercise. Six months before he died he developed marked Cheyne-Stokes respiration, which he had for a week or more, but recovered, and was up again for several months.

I think that some of your readers may be interested in these details. I should be glad to hear the opinions of others on this subject, as I regret I am unable fully to explain the condition myself.—I am, etc.,

London, W.14, April 26th.

RICKARD W. LLOYD.

THE TREATMENT OF INFANTILE PARALYSIS.

SIR,—I have read with much interest the various letters that have recently appeared in the *JOURNAL* on re-education treatment for infantile paralysis. I must, however, point out that Mr. P. B. Roth is not quite right in his letter (April 4th, p. 681) when he states that priority of the methods is to be given to his grandfather, Dr. M. Roth, who published a book on the treatment of paralysis in infancy during 1869. While in no way wishing to detract from the good work done by Dr. M. Roth, in justice it must be stated that his methods were essentially those of the Ling school, who had been treating various forms of paralysis, including infantile paralysis, for many years previously. A case of the disease in question treated by re-education can be found described in the *Archiv für pathologische Anatomie*, 1859, xvi, pp. 177-191, by Dr. M. Eulenburg.—I am, etc.,

London, W.1, April 26th.

EDGAR F. CYRIAX.